APPENDIX B
ENVIRONMENTAL OBJECTIVES THAT APPLY ACROSS ALL SPECIES AND LIFE STAGES

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LIST OF ACRONYMS AND ABBREVIATIONS

°C	degrees Celsius
°F	degrees Fahrenheit
µg/g	micrograms per gram
µg/L	micrograms per liter
7DADM	7-day average of daily maximum temperature
ATP	adenosine triphosphate
Basin Plan	Sacramento and San Joaquin River Basins Water Quality Control Plan
CDFG	California Department of Fish and Game
CDPR	California Department of Pesticide Regulation
CFR	Code of Federal Regulations
ChE	cholinesterase
CRWQCBSDR	California Regional Water Quality Control Board San Diego Region
CTR	California Toxics Rule
CVRWQCB	Central Valley Regional Water Quality Control Board
Delta	Sacramento-San Joaquin Delta
DO	dissolved oxygen
DWR	California Department of Water Resources
ELS	early-life stages
ESA	Endangered Species Act
Hg	mercury
IULT	Incipient Upper Lethal Temperatures
mg/kg	milligram per kilogram
mg/L	milligram per liter
NAWQA	National Water-Quality Assessment
ng/L	nanogram per liter
NMFS	National Marine Fisheries Service
OPP	Office of Pesticide Programs
SEP	Scientific Evaluation Process
SFBRWQCB	San Francisco Bay Regional Water Quality Control Board
SFEI	San Francisco Estuary Institute

SWRCB	State Water Resources Control Board
TDC	TDC Environmental
TMDL	Total Maximum Daily Load
t-TEL	tissue threshold-effect level
USEPA	U.S. Environmental Protection Agency
USFWS	U.S. Fish and Wildlife Service
WDOE	Washington State Department of Ecology
WQC	Water Quality Criterion

1 ENVIRONMENTAL OBJECTIVES AND SUPPORTING RATIONALE FOR VARIABLES THAT APPLY ACROSS ALL SPECIES AND LIFE STAGES

In order to facilitate an integrated understanding of temperature, dissolved oxygen (DO), and contaminants, which are critical to all life stages, the following sections summarize the temperature, DO, and contaminants dynamics and physiological responses broken down in the life stage specific sections.

1.1 Temperature Objectives

1.1.1 Rationale

Salmonid growth and incubation rates, life-stage duration, and metabolic efficiency are directly influenced by water temperature (Quinn 2005). Temperature also has indirect effects on growth rate and incubation rates and success through its interaction with DO concentrations and pathogen activity. Water temperature and developmental rate are tightly and positively correlated (Quinn 2005; Healey 1991); however, beyond certain thresholds, temperature correlates negatively with efficient use of food resources and proper enzymatic functioning. For example, eggs and alevins incubated at temperatures just below their lethal limit produce smaller juveniles than they would at optimal temperatures.

Temperature effects on timing of juvenile emergence and juvenile size at emergence have large impacts on the early life-history and success of developing salmonids. Numerous studies document these sub-lethal effects in different life stages (Quinn 2005; Healey 1991); however, their importance in the overall population dynamics of Chinook salmon populations is not often considered by water and fishery managers.

High water temperatures are a widespread and frequent challenge for several life stages of Central Valley Chinook salmon and steelhead, whereas negative impacts of temperatures near or below low temperature thresholds are uncommon. Several authors have hypothesized that Central Valley populations of Chinook salmon and steelhead may tolerate warmer temperatures than those of other populations (Myrick and Cech 2004). In San Joaquin basin's Tuolumne River, there is limited evidence to support this in *O. mykiss* populations (Farrell et al. 2015), and in general published data do not entirely support the hypothesis.

Temperature-related mortality and habitat-limitation are likely to become even more serious problems for Central Valley salmonids in the future because of global climate change. This makes restoration of salmonid populations in the San Joaquin Valley particularly important as the river and its tributaries drain the highest elevation basins in the lower 48 United States; these watersheds are expected to maintain snowpack (the source of reservoir cold-water pools) further into the future than are watersheds in the northern Central Valley (DWR 2010). San Joaquin Valley Chinook salmon are at the southern edge of their range and access to the coldest waters in this watershed are currently blocked by impassable dams. The dams form reservoirs where water gains heat during the spring and summer before it is released downstream into salmon incubation and rearing habitats. Water management strategies that provide sufficient supplies of cold water for incubating and rearing salmonids are constrained by increasing human demands on water stored in reservoirs and projections of increasing temperatures in the Central Valley (CDFG 2004a, 2004b; Lindley et al. 2007). Reservoir management practices that increase cold-water supplies (e.g., Nickel et al. 2004), those that limit temperature gain of flowing waters (e.g., planting of riparian forests that shade waterways), and restoration of migratory access to colder habitats (NMFS 2009a, 2014) are potential approaches to preserving and expanding incubation and rearing habitat for salmonids in the Central Valley.

In the Central Valley, human ability to actively manage temperatures through reservoir releases diminishes with distance from the reservoir during the late-spring through the mid fall period. Certain riparian and aquatic habitats can limit seasonal temperature gain as water flows to the estuary. Some areas that may have once been used for rearing by juvenile salmonids may no longer be suitable for those functions (even if habitats were restored) because water temperatures have or are expected to increase in those regions as a result of global climate change. Thus, decisions about how and whether to restore salmon rearing habitats at lower elevation are intimately tied to an understanding of thermal limitations of the parr and smolt life stages.

1.1.2 Approach

The Scientific Evaluation Process (SEP) group identified temperature objectives as ranges that are optimal (little or no negative effects), sub-optimal (demonstrably negative, though

perhaps not directly lethal), and detrimental for various salmonid life stages and transitions. In the case of juvenile salmon, temperature objectives were expressed as habitat-specific ranges within a life stage (that reflect the impact of food-availability on temperature response norms) and special attention was given to the metamorphosis of parr to smolt (smoltification) as the success of this transformation is known to be sensitive to elevated temperatures among salmonids.

Estimates of the lethal, sub-optimal, and optimal temperature limits for various life stages of Chinook salmon and steelhead are myriad and variable. Within a species, different life stages have different temperature response curves. Within life stages, variance in estimates of temperature thresholds may result from a combination of factors, including: 1) natural genetic and phenotypic variation among individuals' studied; 2) genetic differences among populations studied; 3) experimental methods and protocols employed by the researchers; and 4) the manner in which experimental data were interpreted and presented in published papers.

The SEP group relied primarily on U.S. Environmental Protection Agency (USEPA; 2003) guidance for temperature effects on Pacific salmon and supplemented that information when newer information and Central Valley-specific studies were available. Except where otherwise noted, temperatures reported here reflect ranges derived from experiments where temperature is held constant throughout the experimental period (i.e., there is no diurnal variation). USEPA (2003) notes that daily average temperatures in the field do not translate directly to static temperatures in a laboratory-diurnal variation in temperatures exposes fish to higher, and potentially injurious, conditions in the field that are not reflected in a situation where temperatures are held constant. Thus, USEPA (2003) recommends use of a 7-day average of daily maximum temperature (7DADM) metric for evaluating temperature impacts on salmonid life stages. Where temperatures in the field exceed those that are optimal, USEPA (2003) proposes a simple conversion of observed (or modeled) temperatures to values that can be compared to static temperatures used in laboratory experiments:

> *When the mean temperature is above the optimal growth temperature, the "midpoint" temperature between the mean and the maximum is the "equivalent" constant temperature. This "equivalent" constant temperature then can be directly*

compared to laboratory studies done at constant temperatures. (19)

In the Stanislaus River, the difference between daily maximum and daily mean temperatures stays roughly constant across seasons, but it does increase with distance downstream from the dam. The difference between the daily maximum and daily mean at the Goodwin Dam gage is approximately 1 degree Celsius (°C) or 1.8 degrees Fahrenheit (°F), while this difference further downstream at the Orange Blossom Bridge gage is approximately 3°C (5.4°F) (Wikert 2014). Thus, the SEP group added approximately 0.5°C (0.9°F) to incubation and early life stage constant temperature thresholds and approximately 1.5°C (2.7°F) to rearing and migration temperature thresholds to provide a 7DADM expression of temperature requirements.

1.1.3 Objectives

1.1.3.1 Chinook Salmon

Life stage specific temperature thresholds were assumed to be the same for spring-run and fall-run Chinook salmon.

1.1.3.1.1 Spawning and Egg Incubation

Adult spawning Chinook salmon temperature needs are generally similar to their eggs. Considerations specific to spawning habitat include temperature triggers for spawning and potential thermal stress that could lead to high rates of prespawn mortality and egg retention. In general, the temperature criteria for eggs are protective of spawning as well as the subsequent egg incubation phase. Salmonid eggs and larvae require cold water to successfully complete incubation. With the construction of impassable dams, Chinook salmon spawning in the San Joaquin Valley became dependent on cold-water storage in reservoirs to provide sufficient cold-water storage to protect their incubating eggs. The accessible supply of cold-water storage limits successful spawning habitat for Chinook salmon populations in the Central Valley in general, and the San Joaquin River basin in particular.

The impact of water temperatures on developing embryos is not well understood. Because the temperature tolerances of fertilized eggs are much lower than those that adult salmon

tolerate, there is concern that developing reproductive tissues exposed to high temperatures may be less viable than those that are formed under cooler temperatures. USEPA (2003) indicates that eggs in holding females exposed to constant temperatures greater than 13°C (55.4°F) suffer reduced viability. Berman (USEPA 1999) found that offspring of adult Chinook salmon that had been held for 2 weeks at temperatures between 17.5°C to 19°C (63.5°F to 66.2°F) had higher pre-hatch mortality and developmental abnormality rates and lower weight than a control group.

USEPA (2003) found that constant temperatures between 4°C and 12°C (39.2°F and 53.6°F) result in good egg survival and that a narrower range (6°C to 10°C [42.8°F to 50°F]) is optimal; a 7DADM of less than 13°C (55.4°F) is recommended (Table B-1). In a review, the U.S. Fish and Wildlife Service (USFWS; 1999, cited by Myrick and Cech 2004) concluded that temperature-related egg mortality in Chinook salmon increased at temperatures above 13.3°C (55.9°F) and this is the limit applied in most regulatory arenas (e.g., NMFS 2009a; SWRCB Order 90-05). A review of research on different populations of Chinook salmon from within and outside of the Central Valley indicated that temperatures between 6°C and 12°C (42.8°F and 53.6°F) were optimal for Central Valley Chinook salmon (Myrick and Cech 2004).

Table B-1 identifies the optimal, sub-optimal, and detrimental temperature conditions for Chinook salmon spawning and egg incubation.

Table B-1

Temperature Objectives for Chinook Salmon Spawning and Egg Incubation

Spatial Extent (Habitat Type)	Temporal Extent	Condition	Range (Metric)
Gravel	**Fall-run:** Late October to March **Spring-run:** Late August to March	Optimal	6°C to 12°C (42.8°F to 53.6°F) (Daily Average)
			< 12.5°C to 13°C (54.5°F to 55.4°F)(7DADM)
		Sub-optimal	4°C to 6 °C (Daily Average)
			12°C to 13.3°C (53.6°F to 55.9°) (Daily Average)
			12.5°C to 13.8°C (54.5°F to 56.8°F) (7DADM)
		Detrimental	> 13.3°C (55.9°F) (Daily Average)
			> 13.8°C (56.8°F) (7DADM)

Notes:
"<" = greater than
"<" = less than
°C°F = degrees Fahrenheit
7DADM = 7-day average of daily maximum temperature

1.1.3.1.2 Juvenile Rearing and Migration

Temperatures that produce mortality among Pacific salmon depend, to some extent, on acclimation temperatures—higher acclimation temperatures produce higher Incipient Upper Lethal Temperatures (IULT; Myrick and Cech 2004). Various sources indicate an IULT for Chinook salmon in the range of 24°C to 25°C (75.2°F to 77°F) (Myrick and Cech 2004). Baker et al. (1995) found that Central Valley Chinook salmon had an IULT between approximately 22°C to 24°C (71.6°F to 75.2°F).

Negative sub-lethal effects (those that may increase susceptibility to other mortality mechanisms) begin to occur at temperatures lower than the IULT. In the laboratory, when fish have access to full rations, growth of juvenile salmonids increases with temperature up to fishes' physiological limits; however, when food supply is limited (as it often is under normal conditions in the field) optimal and sub-optimal growth and even mortality occur at lower temperatures. For example, Mesa et al. (2002) detected increased levels of heat shock proteins (an indicator of stress) after several hours of exposure to 20°C (68°F) for Columbia River fall-run Chinook salmon. Among juvenile fall-run Chinook salmon from California's Central Valley population, Marine and Cech (2004) found decreased growth, reduced smoltification success, and impaired ability to avoid predation at temperatures above 20°C (68°F). They also reported that fish reared at temperatures from 17°C to 20°C (62.6°F to 68°F) experienced increased predation relative to fish raised at 13°C to 16°C (55.4°F to 60.8°F), although they found no difference in growth rate among fish reared in these two temperature ranges. The finding of decreased performance at temperatures above 17°C (62.6°F) is consistent with several studies that suggest, when food supplies are not super-abundant, optimal growth and survival among Chinook salmon occurs at temperatures somewhat lower than 17°C (62.6°F). USEPA (2003) identifies constant temperatures of identifies constant temperatures of 10°C to 17°C (50°F to 62.6°F) (and a 7DADM less than 18°C [64.4°F]) as being optimal conditions for juvenile Chinook salmon when food supplies are limiting. USEPA (2003) recommends 16°C (60.8°F) 7DADM as a maximum criterion to: 1) safely protect juvenile salmon and trout from lethal temperatures; 2) provide upper optimal conditions for juvenile growth under limited food during the period of summer maximum temperatures and optimal temperatures for other times of the growth season; 3) avoid temperatures where juvenile salmon and trout are at a competitive disadvantage

with other fish; 4) protect against temperature induced elevated disease rates; and 5) provide temperatures that studies show juvenile salmon and trout prefer and are found in high densities. Based on this recommendation, 16°C (60.8°F) 7DADM or less has been established as the optimal water temperature for juvenile rearing and migration in the river channel.

As indicated, the temperatures that can be tolerated by rearing juvenile Chinook salmon depend to a great extent on food availability. USEPA (2003) indicates that, when food supplies are "unlimited" temperatures from 13°C to 20°C (55.4°F to 68°F) (constant) may be optimal. Recent studies on Central Valley Chinook salmon rearing on inundated floodplains reveal excellent survival and growth rates at even higher temperatures. Growth and survival have been recorded at temperatures as high as approximately 25°C (77°F) (Katz unpublished data; Jeffres unpublished data). The increased tolerance for high temperatures in these fish is believed to be related to the relatively high abundance of high quality food available to Chinook salmon rearing on floodplains and suggests that, when food is not limiting, Chinook salmon can tolerate and even thrive in the wild at temperatures approaching the physiological limits observed in the laboratory (i.e., IULT). As a result, the SEP group assumed that, following successful restoration of floodplain habitats (and during periods when juvenile Chinook salmon actually occupy inundated floodplains), rearing Chinook salmon juvenile salmon could survive temperatures approaching 25°C (77°F). For example, the life-history timing and productivity objectives for both spring and fall-run Chinook salmon salmon could survive temperatures approaching 25°C (77°F) for limited periods of time. Based on these distinctions, temperatures greater than 25°C were established a detrimental for salmon rearing on long-inundation floodplains only. However, the SEP group also recognizes that exposure to such warm water temperatures greatly increases disease risk, and stress from other water quality factors (e.g., DO or contaminants) likely reduces thermal tolerance. When Chinook salmon are not in habitats that support super-abundant food resources (e.g., in mainstem channel habitats), lower temperatures are required to avoid negative sub-lethal effects.

Elevated water temperatures can inhibit the parr-smolt metamorphosis (smoltification) in salmonids. Chinook salmon can smolt at temperatures ranging from 6°C to 20°C (42.8°F to 68°F) (Myrick and Cech 2004). However, salmon that smolt at higher temperatures (greater than 16°C (60.8°F) tend to display impaired smoltification patterns and reduced saltwater

survival (Myrick and Cech 2004). Marine and Cech (2004) found that Central Valley Chinook salmon rearing in temperatures greater or equal to 20°C (68°F) suffered altered smolt physiology, and other studies from within this ecosystem suggest that negative effects of temperature on the parr-smolt transition may occur at temperatures less than 20°C (68°F). Richter and Kolmes (2005) cite two studies that indicated negative impacts on Chinook salmon smoltification success at temperatures greater than 17°C (62.6°F) and USEPA (2003) indicates that smoltification impairment may occur at temperatures between 12°C to 15°C (53.6°F to 59°F).

Table B-2 identifies the optimal, sub-optimal, and detrimental temperature conditions for juvenile Chinook salmon rearing and migration.

Table B-2
Temperature Objectives for Juvenile (Fry, Parr, and Smolt) Chinook Salmon Rearing and Migration

Spatial Extent (Habitat Type)	Temporal Extent	Condition[1]	Range (Metric)
Channel	**Fall-run:** Last week of January to the 2nd week of June **Spring-run:** First week of January to the 2nd week of June	Optimal	6°C to 16°C (42.8°F to 60.8°F) (7DADM)
		Sub-optimal	17°C to 20°C (62.6°F to 68°F) (7DADM)
		Detrimental	> 20°C (68°F) (7DADM)
Off-Channel – (Short Inundation)		Optimal	10°C to 18°C (50°F to 64.4°F) (7DADM)
		Sub-optimal	18°C to 20°C (64.4°F to 68°F) (7DADM)
		Detrimental	> 20°C (68°F) (7DADM)
Inundated Floodplain – (Long Inundation)		Optimal	10°C to 18°C (50°F to 64.4°F) (7DADM)
		Sub-optimal	18°C to 25°C (7DADM)
		Detrimental	> 20°C (68°F) (7DADM)

Notes:
[1] These temperatures apply all along the juvenile migratory corridor. Because water temperatures are expected to increase as water travels downstream during warmer months, temperatures measured or modeled upstream that are at or near the limit of a given range would be expected to exceed that range further downstream. Thus, temperatures at the high end of the sub-optimal range that are measured or modeled in upstream locations indicate potentially detrimental temperature conditions further downstream, including into the San Joaquin mainstem.
">" = greater than
°C°F = degrees Fahrenheit
7DADM = 7-day average of daily maximum temperature

1.1.3.1.3 Adult Migration

High water temperatures can lead to direct mortality and indirect loss of fitness for migrating salmon. The IULT may be as low as 21°C to 22°C (69.8°F to 71.6°F) for both adult Chinook salmon and steelhead during migration (USEPA 1999, 2003; Richter and Kolmes 1995). Swimming performance is reduced at temperatures greater than 20°C (68°F) (USEPA 2003). High water temperatures also facilitate infection among migrating adult salmonids (Noga 1996); USEPA (2003) identifies an elevated risk of infection at temperatures above 14°C (57.2°F) and a high risk of infection at temperatures greater than 18°C (64.4°F). Unlike juvenile salmon, the response of adult salmon to high temperatures is not related to food availability—adult salmon typically do not feed during their freshwater migration or holding period.

Water temperatures below the IULT may also impede spawning migrations. Prolonged exposure to temperatures greater than 17°C (62.6°F) reduce fitness during migration due to cumulative stresses (USEPA 2003); in fact, McCullough et al. (2001), writes: "Migration blockages, susceptibility to disease, impaired maturation process, increases to stress parameters, reduced efficiency of energy use, and reduced swimming performance were all cited [by MacDonald in press] as potentially serious hazards as daily mean temperatures exceed 62.6°F (17°C)" (p. 9). Higher temperatures may produce acute distress.

Williams (2006) reported that salmon returning to the Stanislaus River in 2003 endured water temperatures greater than 21°C (69.8°F) on their migration; however, there is no indication that these fish spawned successfully or that they produced viable offspring. Williams (2006) reported that migrating Sacramento River fall-run Chinook adult salmon appeared to avoid temperatures greater than approximately 19°C (66.2°F), an observation consistent with reports for Chinook salmon from other watersheds (Richter and Kolmes 2005). Many sources recommend maintaining temperatures less than 20°C to 21°C (68°F to 69.8°F) to prevent direct impairment of Chinook salmon migrations (Richter and Kolmes 2005; USEPA 1999, 2003).

Table B-3 identifies the range in temperatures associated with optimal, sub-optimal, and detrimental conditions for Chinook salmon adult migration and holding.

Table B-3

Temperature Objectives for Chinook Salmon Adult Migration and Holding

Spatial Extent (Habitat Type)	Temporal Extent	Condition	Range (Metric)
Main Channel	**Fall-run:** Late September to December **Spring-run:** March to July (Migration); March to September (Holding)	Optimal	8°C to 14°C (46.4°F to 57.2°F) (Daily Average)
			9.5°C to 15.5°C (49.1°F to 59.9°F) (7DADM)
		Sub-optimal	14°C to 19°C (57.2°F to 66.2°F) (Daily Average)
			15.5°C to 20.5°C (59.9°F to 68.9°F) (7DADM)
		Detrimental	> 18°C (64.4°F) (weekly mean)
			> 19°C (66.2°F) (Daily Average)
			> 20.5°C (68.9°F) (7DADM)
			> 22°C (71.6°F) (instantaneous)

Notes:
">" = greater than
°C°F = degrees Fahrenheit
7DADM = 7-day average of daily maximum temperature

1.1.3.2 Steelhead

1.1.3.2.1 Spawning and Egg Incubation

As with Chinook salmon, adult spawning steelhead temperature needs are generally similar to their eggs. Considerations specific to spawning habitat include temperature triggers for spawning and potential thermal stress that could lead to high rates of prespawn mortality and egg retention. In general, the temperature criteria for eggs are protective of spawning as well as the subsequent egg incubation phase. *O. mykiss* eggs and larvae require cold water to successfully complete incubation. With the construction of impassable dams, *O. mykiss* eggs incubating in the San Joaquin Valley became dependent on cold-water storage in reservoirs. The accessible supply of cold-water storage limits successful spawning habitat for *O. mykiss* populations in the southern Central Valley. There is a serious lack of peer-reviewed studies on the temperature tolerances of Central Valley anadromous *O. mykiss* eggs, and additional study of temperature impacts on this species' eggs is needed (Myrick and Cech 2004). Optimal incubation temperatures for steelhead occur in a narrower range than those for Chinook salmon. Indeed, Myrick and Cech (2004) warned against managing water temperatures for the upper end of the Chinook salmon thermal tolerance range in waterways

and during periods when steelhead are also incubating because incubating steelhead cannot tolerate such high temperatures. Richter and Kolmes (2005) concluded that egg mortality increased as incubation temperatures exceeded 10°C (50°F) and substantial mortality may occur when temperatures exceed 13.5°C to 14.5°C (56.3°F to 58.1°F). Based on experience at hatcheries in the Central Valley, optimal incubation temperatures appear to be in the 7°C to 10°C (50°F) range (Myrick and Cech 2004). California's steelhead management plan (McEwan and Jackson, 1996) suggests a slightly higher temperature range (from 9°C to 11°C [48.2°F to 51.8°F).

Table B-4 identifies optimal, sub-optimal, and detrimental temperature conditions for steelhead spawning and egg incubation.

Table B-4
Temperature Objectives for Steelhead Spawning

Spatial Extent (Habitat Type)	Temporal Extent	Condition	Range (Metric)
Gravel	December to June	Optimal	7°C to 10°C (44.6°F to 50°F) (Daily Average)
			10.5°C (50.9°F) (7DADM)
		Sub-optimal	4°C to 6.9°C (39.2°F to 44.4°F) (Daily average)
			10°C to 13.5°C (50°F to 56.3°F) (Daily Average)
			10.5°C to 14.0°C (50.9°F to 57.2°F) (7DADM)
		Detrimental	> 13.5°C (56.3°F) (Daily Average)
			> 14.0°C (57.2°F) (7DADM)

Notes:
">" = greater than
°C°F = degrees Fahrenheit
7DADM = 7-day average of daily maximum temperature

1.1.3.2.2 Juvenile Rearing and Migration

Laboratory studies show that incipient lethal temperatures for juvenile steelhead occur in a range between 27.5°C to 29.6°C (81.5°F to 85.3°F), depending on acclimation temperatures (Myrick and Cech 2005). Optimal temperatures for steelhead juvenile growth occur between 15°C to 19°C (59°F to 66.2°F) (Moyle 2002; Richter and Kolmes 2005). Temperature also mediates the impact of competition between species. For example, steelhead juveniles suffer adverse impacts of competition with pikeminnow at temperatures greater than 20°C (68°F),

though no competitive impact is detectable at lower temperatures (Reese and Harvey 2002).

Table B-5 identifies optimal, sub-optimal, and detrimental temperature conditions for steelhead juvenile rearing.

Table B-5
Temperature Objectives for Steelhead Juvenile Rearing

Spatial Extent (Habitat Type)	Temporal Extent	Condition[1]	Range (Metric)
Mainstem	January to December (i.e., year round)	Optimal	15°C to 19°C (59°F to 66.2°F) (Daily Average)
			16.5°C to 21.5°C (61.7°F to 70.7°F) (7DADM)
		Sub-optimal	20°C to 25°C (68°F to 77°F) (Daily Average)
			21.5°C to 26.5°C (70.7°F to 79.7°F) (7DADM)
		Detrimental	> 25°C (77°F) (Daily Average)
			26.5°C (79.7°F) (7DADM)
			> 27.5°C (81.5°F) (Instantaneous)

Notes:
[1] These temperatures apply all along the juvenile migratory corridor. Because water temperatures are expected to increase as water travels downstream during warmer months, temperatures measured or modeled upstream that are at or near the limit of a given range would be expected to exceed that range further downstream. Thus, temperatures at the high end of the sub-optimal range that are measured or modeled in upstream locations indicate potentially detrimental temperature conditions further downstream, including into the San Joaquin mainstem.
">" = greater than
°C°F = degrees Fahrenheit
7DADM = 7-day average of daily maximum temperature

Steelhead may be particularly sensitive to high temperatures during the smoltification process. USEPA (2003) indicates that temperatures greater than 12°C (53.6°F) inhibit steelhead metamorphosis into smolts. Richter and Kolmes (2005) and USEPA (1999) cited studies that present a range of temperatures, between 11°C and 14°C (51.8°F and 57.2°F) that may inhibit steelhead smoltification. Myrick and Cech (2005) cautioned that smolting steelhead in the Central Valley must experience temperatures less than 11°C (51.8°F) to successfully complete this metamorphosis. The critical temperature at which smoltification becomes inhibited may vary from run-to-run (Richter and Kolmes 2005).

Table B-6 identifies the optimal, sub-optimal, and detrimental temperature conditions for

juvenile steelhead smoltification.

Table B-6

Temperature Objectives for Steelhead Juvenile Migration (Smoltification)

Spatial Extent (Habitat Type)	Temporal Extent	Condition	Range (Metric)
Main Channel	December to March	Optimal	11°C (51.8°F) (Weekly Average)
			12.5°C (54.5°F) (7DADM)
		Detrimental	> 11°C (51.8°F) (Weekly Average; i.e., detrimental if necessary temperature is not achieved during appropriate annual window)
			> 12.5°C (54.5°F) (7DADM)

Notes:
"<" = greater than
°C°F = degrees Fahrenheit
7DADM = 7-day average of daily maximum temperature

1.1.3.2.3 Adult Migration and Holding

The IULT may be as low as 22 °C (71.6°F) for migrating steelhead (USEPA 1999; Richter and Kolmes 1995). Although steelhead have been known to migrate in most months of the year, they are mostly present from mid fall to early spring (Hallock 1961; Harvey 1995; McEwan 2001) when temperatures are generally well-below the lethal threshold. For purposes of this report, the SEP group has assumed that temperatures, which are acceptable to migrating Chinook salmon adults are also acceptable to migrating steelhead adults.

Table B-7 provides the optimal, sub-optimal, and detrimental temperature conditions for adult steelhead migration.

Table B-7

Temperature Objectives for Steelhead Migration, Holding, and Post-spawning Adults (Kelts)

Spatial Extent (Habitat Type)	Temporal Extent	Condition	Range (Metric)
Main Channel	Mid-September to Mid-May	Optimal	8°C to 14°C (46.4°F to 57.2°F) (Daily Average)
			9.5°C to 15.5°C (49.1°F to 59.9°F) (7DADM)
		Sub-optimal	

			14°C to 19°C (57.2°F to 66.2°F) (Daily Average)
			15.5°C to 20.5°C (59.9°F to 68.9°F) (7DADM)
		Detrimental	> 18°C (64.4°F) (Weekly average)
			> 19°C (66.2°F) (Daily Average)
			20.5°C (68.9°F) (7DADM)
			> 22°C (71.6°F) (Instantaneous)

Notes:
">" = greater than
°C°F = degrees Fahrenheit
7DADM = 7-day average of daily maximum temperature

1.2 Dissolved Oxygen Objectives

1.2.1 Rationale

Adequate concentrations of DO in water are critical for salmon and steelhead survival. In freshwater streams, hypoxia can impact the growth and development of salmon and steelhead eggs, alevins, and fry, as well as the swimming, feeding, and reproductive ability of juveniles and adults. If salmonids are exposed to hypoxic conditions for too long, mortality can result (Carter 2005). Without achieving some combination of optimal and/or sub-optimal environmental objectives for DO described below, the biological objectives for Chinook salmon and steelhead most certainly will not be met.

1.2.2 Approach

The SEP group relied on DO criteria established by the USEPA (1986) and the Central Valley Regional Water Quality Control Board (CVRWQCB; 2011) as well as relevant technical literature (e.g., WDOE 2002) to identify DO objectives that are optimal (no negative effects), sub-optimal (observably negative, though not significantly harmful), and detrimental (clearly harmful) ranges for various salmonid life stages and/or transitions. The approach the SEP group used to translate available information on impairment levels into optimal, sub-optimal, and detrimental objectives is shown in Table B-8.

Table B-8
Recommended Cold-Water Species DO Levels for Spawning, Egg Incubation, and Larval Life Stages

Level of Impairment to Embryo and Larvae Stages	Water Column Minimum Average Concentration	Intra-Gravel Minimum Average Concentration	Optimal, Sub-Optimal, Detrimental[1]
No production impairment	11 mg/L	8 mg/L	Optimal
Slight production impairment	10 mg/L	7 mg/L	Sub-optimal
Slight production impairment	9 mg/L	6 mg/L	Sub-optimal
Moderate production impairment	8 mg/L	5 mg/L	Detrimental
Severe production impairment	7 mg/L	4 mg/L	Detrimental
Limit to avoid acute mortality	6 mg/L	3 mg/L	Detrimental

Notes:
[1] Relationship of recommended DO levels to optimal, sub-optimal, and detrimental levels identified by the SEP group
Table adapted from USEPA 1986
mg/L = milligram per liter

The criteria established by the USEPA and Central Valley Regional Water Quality Control Board (CVRWQCB) covered cold water species in one category; separate criteria for Chinook salmon and steelhead were not provided. This blanket approach of protecting salmon and steelhead with one set of DO criteria is supported by the available literature, and as such, the SEP group followed that approach. While it was not necessary to have species-specific DO objectives, life stage-specific ones are needed because dissolved oxygen requirements for eggs and larvae slightly differ from those for juveniles and adults.

The following summaries of egg incubation mortality through hatching, incubation growth rates, juvenile rearing and migration, and adult migration and holding provide life-stage specific rationale for the DO objectives presented in Section 1.2.3.

1.2.2.1 Egg Incubation Mortality through Hatching (from WDOE 2002)

At favorable incubation temperatures mortality rates should be expected to remain less than 1% at a concentration of 9 milligrams per liter (mg/L) or greater, less than 2% at a concentration of 7 mg/L, and between 2% and 6% percent at a concentration of 6 mg/L. While mean oxygen concentrations over the development period below 6 mg/L are sometimes associated with significant increases in mortality rates, the overall pattern is for mortality rates and the occurrence of abnormalities to remain low (less than 7%) at concentrations above 4 mg/L. Survival rates at oxygen concentrations below 4 mg/L are highly variable. While mortality rates were low (4% to 7%) in some studies, they ranged

from 25% to 100% in others. All tests at concentrations below 1.7 mg/L resulted in 100% mortality. While mortality rates related to low oxygen concentrations remain relatively minor at favorable incubation temperatures (averages below 11°C [51.8°F]), they increase rather substantially at temperatures that are warmer than ideal. In warmer waters (13.4°C [56.1°F]) even a decrease from 11 to 10 mg/L would be associated with causing a 4% reduction in survival through hatching. A decrease to 7 mg/L would be associated with a 19% reduction in survival. An important point to recognize is that in the laboratory studies the developing alevin did not need to push their way up through gravel substrate as would wild fish. The studies above focused on survival through hatching and did not consider this rather substantial final act for emerging through the redds. Optimal fitness will likely be required for optimal emergence in the natural environment, and the metabolic requirements to emerge would be expected to be substantial. Thus higher oxygen levels may be needed to fully protect emergence than to just fully support hatching alone.

1.2.2.2 Incubation Growth Rates (from WDOE 2002)

Any decrease in the mean oxygen concentration during the incubation period appears to directly reduce the size of newly hatched salmonids. At favorable incubation temperatures the level of this size reduction, however, should remain slight (2%) at mean oxygen concentrations of 10.5 mg/L or more and still remain below 5% at concentrations of 10 mg/L or more. At 9 mg/L, the size of hatched fry would be reduced approximately 8%. Mean concentrations of 7mg/L and 6 mg/L would be expected to cause 18 and 25% reductions in size.

1.2.2.3 Juvenile Rearing and Migration

Salmonids may be able to survive when DO concentrations are low (less than 5 mg/L), but growth, food conversion efficiency, and swimming performance will be adversely affected (Bjornn and Reiser 1991). Davis (1975) reviewed numerous studies and reported no impairment to rearing salmonids if DO concentrations averaged 9 mg/L, while at oxygen levels of 6.5 mg/L "the average member of the community will exhibit symptoms of oxygen distress," and at 4 mg/L a large portion of salmonids may be affected. WDOE (2002) concludes that a monthly or weekly average concentration of 9 mg/L, and a monthly average of the daily minimum concentrations should be at or above 8.0 to 8.5 mg/L to have a negligible effect (5% or less) on growth and support healthy growth rates. USEPA (1986)

states that due to the variability inherent in growth studies, the reductions in growth rates seen above 6 mg/L are not usually statistically significant, while reductions in growth at DO levels below 4 mg/L are considered severe. WDOE (2002) recommended that DO levels below 5.0 to 6.0 mg/L should be considered a potential barrier to the movement and habitat selection of juvenile salmonids. Given that recommendation, DO levels below 6.0 mg/L have been established as detrimental for juvenile salmon.

1.2.2.4 Adult Migration and Holding

WDOE (2002) reported that DO concentrations above 8 to 9 mg/L are needed for maximum swimming performance and concentrations below 5 to 6 mg/L elicited avoidance. Hallock et al. (1970) found that adult Chinook salmon migrating up the San Joaquin River avoided DO concentrations below 5 mg/L. DO concentrations above 8 mg/L were assumed to represent optimal conditions and concentrations below 6 mg/L were detrimental. Between 6 and 8 mg/L was identified as sub-optimal for migrating and holding adults.

1.2.3 Objectives

DO objectives are provided in Tables B-9 and B-10 by the following two life stage groupings:

- Spawning adults, eggs, and larvae
- Rearing and emigrating fry and juveniles and immigrating and holding adults

These groupings are consistent with the USEPA and CVRWQCB DO criteria and the supporting technical literature. Anadromous salmonid eggs and larvae are more sensitive to low DO concentrations than rearing juveniles and adults that are immigrating or holding.

Table B-9
DO Objectives for Chinook Salmon and Steelhead Spawning and Egg Incubation

Spatial Extent (Habitat Type)	Temporal Extent	Condition	Range (Metric)
Gravel (Measurement must occur in gravel, not water column)	**Fall-run:** Late October to March	Optimal	> 8 mg/L (Daily Minimum)
		Sub-optimal	6 to 8 mg/L (Daily Minimum)
	Spring-run: Late August to March		
	Steelhead: December to June		

		Detrimental	< 6 mg/L (Daily Minimum)

Notes:
">" = greater than
"<" = less than
mg/L = milligram per liter

Table B-10
DO Objectives for Chinook Salmon and Steelhead Fry/Juveniles and Migrating and Holding Adults

Spatial Extent (Habitat Type)	Temporal Extent	Condition	Range (Metric)
River Channel or Floodplain (Water column measurement)	**Fall-run:** Last week of January to 2nd week of June (fry/juveniles) Late September to December (migration and holding) **Spring-run:** January to December (i.e., year-round) (fry/juveniles) March to July (migration) March to September (holding) **Steelhead:** January to December (i.e., year-round) (fry/juveniles) Mid-September to mid-May (migration and holding)	Optimal	> 8 mg/L (Daily Minimum)
		Sub-optimal	6 to 8 mg/L (Daily Minimum)
		Detrimental	< 6 mg/L (Daily Minimum)

Notes:
">" = greater than
"<" = less than
mg/L = milligram per liter

1.3 Contaminant Objectives

1.3.1 Rationale

The Stanislaus River, between Goodwin Dam and Caswell State Park, has been identified as

being impaired on the USEPA Clean Water Act Section 303(d) list for not meeting water quality standards since the early 1990s. The pollutants or stressors that have been identified to cause the impairments are: diazinon, chlorpyriphos, Class A pesticides (e.g., organochlorines, DDT, and legacy pesticides), unknown toxicity, mercury, and temperature (USEPA 2011). In addition, mercury and selenium have been identified as impairing beneficial uses in the San Joaquin River, the Sacramento-San Joaquin Delta (Delta), and the San Francisco Bay, which are downstream salmonid rearing and migratory habitats (SWRCB 2010; USEPA 2011). Beneficial uses that are not being supported include: cold freshwater habitat; migration; spawning, reproduction and early development; and warm freshwater habitat. Other contaminants that were evaluated, but were found not to exceed water quality standards included ammonia, arsenic, cadmium, and nickel (SWRCB 2010).

The large majority of currently available spawning habitat and subsequent rearing habitat in the Stanislaus River is below Knights Ferry (ESA 2013), and this reach coincides with increased amounts of anthropogenic disturbances, primarily agricultural and urban development. In a review of toxicity monitoring data conducted in California, Anderson and others (2011) found that sites located near agriculture and urban areas had statistically greater occurrences of toxicity in water and sediment samples than near undeveloped areas. In all, 51% and 45% of the streams, rivers, canals, and lakes monitored from 2001 to 2010 had some toxicity in the water column and sediment, respectively. Toxicological effects can range from sublethal endpoints to full organism mortality. Using correlation analyses and toxicity identification evaluations, Anderson et al. (2011) determined that the vast majority of toxicity was caused by pesticides (e.g., insecticides, herbicides, and fungicides). However, pesticides were not the cause of all toxicity, and some other contaminants that were identified included metals and ammonia.

The CVRWQCB has recently developed a control program and adopted water quality objectives for diazinon and chlorpyriphos in the Central Valley (CVRWQCB 2014), so the implementation of the program should reduce the adverse impacts of these two constituents. However, the use of organophosphate pesticides like diazinon and chlorpyriphos have declined in California since the mid-1990s, and USEPA actions resulted in the phase out of these two pesticides for urban use in the early 2000s (Spurlock and Lee 2008). Much of the pesticide use has shifted to pyrethroids, especially for urban use, and in 2006 pyrethroids

accounted for greater than 40% of the insecticide registrations in California. Pyrethroids have been identified as causing much of the surface water and sediment toxicity in California (Anderson et al. 2011). More recently, the use of the systemic pesticides neonicotinoids has increased, and their use has been implicated in global declines of some wildlife (Gibbons et al. 2014; Mason et al. 2013). Current use pesticides are ever changing, and this makes it difficult for regulatory agencies to control the adverse effects that these contaminants create.

Mercury and selenium both occur naturally in the environment; however, anthropogenic activities have resulted in elevated concentrations in surface waters that are a detriment to aquatic life. For centuries, the smelting of large quantities of ore has contributed to the emissions of trace metals worldwide (Nriagu 1996). Recently, mercury water quality impairments in California have been linked to local and international industrial emissions (SFEI 2001; USEPA 2008). Extensive historical mining in California contributed to heavy metal emissions, as well abandoned mine waste material continues to pollute Central Valley waterbodies (Alpers and Hunerlach 2000; Domagalski 2001; and USEPA 2006). Oil refining and agricultural irrigation have contributed to selenium contamination in the San Francisco Bay and the San Joaquin River watershed, respectively (McCarthy and Grober 2001; Presser and Luoma 2006, 2013). In addition, urban storm water runoff has been shown to be a major source of metals to California surface waters (CRWQCBSDR 2007; SFBRWQCB 2007; TDC 2004).

The following sub-sections will describe the three major contaminants (pesticides[1], mercury, and selenium) that have been identified as impairing beneficial uses in the Stanislaus River and downstream migratory corridor. The descriptions of each contaminant will include the general background and the toxicological effects of each contaminant to fish, with emphasis on salmonids where available. If other contaminants or toxins are identified to impeded Chinook salmon and steelhead recovery in the San Joaquin River basin, then their impacts can be evaluated in the future.

1.3.1.1 Pesticides

Fish are not the target organisms of the pesticides; however, pesticides have been found to

[1] The pesticide section will include a discussion on copper effects because copper is widely used as pesticide (e.g., fungicide, herbicide, and antifouling paint).

cause adverse impacts to fish in surface waters. For example, in a review of Central Valley toxicity data, Markiewicz et al. (2012) found that the fish species tests, *Pimephales promelas*, had a higher frequency of toxicity than the other species, *Ceriodaphnia dubia* (invertebrate) and *Selenastrum capricornutum* (algal). Samples were toxic to fish in 62% of the tests versus 49% for invertebrates and 40% for algae. Similar to the statewide survey of Anderson and others (2011), pesticides were found to be the primary cause of toxicity in the Central Valley (Markiewicz et al. 2012). Importantly, salmonids generally tend to be more sensitive to chemical stressors than many other species of fish; and, if other freshwater fish are killed by use of pesticides, then it is likely that salmonids have also died (NMFS 2012).

Moreover, the life-history strategies salmonids evolved to rely on exposes them to higher risks from contaminants. For example, juvenile salmonids typically occupy and rely on shallow freshwater habitats (e.g., floodplains, off-channel, and low flow alcoves) during critical rearing and migratory life-history periods. These near-shore, low flow habitats are expected to have higher pesticide loading and concentrations, which subject developing salmonids to higher exposures to pesticides in their preferred habitats (NMFS 2008, 2009b, and 2011). Even if salmonids can avoid the elevated concentrations of contaminants in these areas, salmonids may be adversely impacted by not benefitting from the uses these habitats provide (e.g., food and cover).

Typically, adult organisms will have a lower risk of mortality to contaminants than the sensitive larval fish used for toxicity tests. As a result, toxicity tests with larval fish could overestimate the mortality that might occur to adult salmonids. However, pre-spawn adult salmonids are likely less tolerant to chemical stressors because they have used most of their accumulated fat stores for gamete production (NMFS 2008, 2010, and 2013). It is probable that the some pre-spawn returning adults will die as a result of short-term exposures to pesticides, especially when subjected to additional stressors like elevated temperatures. Additionally, pre-spawn mortality can be cause by other contaminants. For example, metals and petroleum hydrocarbons likely contributed to pre-spawn mortality of Coho salmon in urban streams in Washington State (Scholz et al. 2011). Pre-spawn mortality is a particularly important factor in the recovery of salmonid populations with low abundance because every adult is crucial to the population's viability (NMFS 2013).

While direct mortality is an obvious detriment to salmonid populations, many sublethal effects of pesticide can also contribute to population declines. Sublethal toxicant exposure often eliminates the performance of fish behaviors, such as predator avoidance, orientation, reproduction, kin recognition, etc. that are essential to fitness and survival in natural ecosystems (Potter and Dare 2003; Scott and Sloman 2004). The most commonly observed links with behavioral disruption include cholinesterase (ChE) inhibition, altered brain neurotransmitter levels, sensory deprivation, and impaired gonadal or thyroid hormone levels (Scott and Sloman 2004). For example, Scholz and others (2000) concluded that olfactory disruption by anti-cholinesterase neurotoxins reduced Chinook salmon anti-predator responses from short-term, sublethal exposures to diazinon. As well, they also concluded that 24-hour exposures to diazinon likely increased the straying of the adult hatchery Chinook salmon over the control group. Furthermore, juvenile salmonids exposed to pesticides during development may fail to imprint to their natal waters, which can lead to increased adulthood straying (NMFS 2009b).

Additional evidence of the sublethal effects of pesticides on fish populations have been demonstrated though reproduction experiments. For example, the pyrethroid insecticide cypermethrin inhibited male Atlantic salmon from detecting and responding to the reproduction priming pheromone prostaglandin, which is released by ovulating females (Moore and Waring 2001). The males exposed to cypermethrin did not respond to prostaglandin with the expected increased levels of plasma sex steroids and expressible milt. In addition, zebrafish exposed to low concentrations (96-hour LC5) of deltamethrin and Achook (a synthetic pyrethroid and a neem based pesticide, respectively) resulted in significant reductions (54% and 18%, respectively) in female fecundity when compared to the controls (Sharma and Ansari 2010). Additionally, both of the studies found that exposures to pesticides decreased the abundance of hatchlings. The percentage of unhatched fertilized eggs increased in adult zebrafish exposures, and the number of unfertilized eggs increased in salmon egg and milt exposures (Sharma and Ansari 2010; Moore and Waring 2001). Furthermore, the disruption of spawning synchronization could also result in an increase in the number of unfertilized eggs (NMFS 2009b).

Herbicide pesticides also have been shown to reduce fish's ability to perform necessary physiological activities. For example, Waring and Moore (1996) observed that

concentrations of the herbicide atrazine that showed no lethal effects to Atlantic salmon in freshwater resulted in physiological stress and increased mortality once the fish were exposed to seawater. Subsequent investigations determined that sublethal concentrations of atrazine can reduce $Na^+ K^+$ ATPase activity and the ability of salmon to osmoregulate (Moore and Fewings 2003). Nieves-Puigdoller and others (2007) found similar disruptions in osmoregulation as well as other endocrine disruption, however at higher concentrations of atrazine. Other investigations have concluded that another herbicide, trifluralin, can cause vertebral deformities, which would likely also result in the eventual mortality from predators or reduced prey capture (NMFS 2012). Because pesticides are developed and used for multiple target organisms (e.g., plants, invertebrates, and vertebrates), their mechanisms of action are very diverse. This results in a multitude of ways that pesticides can affect salmonid physiology, biochemistry, and behavior, and subsequently, many different life stages of salmonids can be adversely impacted.

Copper compounds are also often used as herbicides in addition to other types of pesticides, and copper is one of the most widely applied pesticides in the Central Valley (Johnson et al. 2010). Additionally, copper is a naturally occurring trace element, and non-pesticide related anthropogenic activities have increased copper pollution to surface waters. For example, other sources of copper to surface waters include: urban runoff (e.g., vehicle brake pads, architectural features, and industrial uses), mining waste, soil erosion, etc. (CVRWCB 2002; TDC 2004). Extreme cases of copper and other heavy metal contamination resulted in acid mine drainage that contributed to fish kills and significant declines in Chinook salmon and steelhead populations in the Sacramento River from the 1960s to the 1980s (CVRWQCB 2002). Heavy metal pollution from the Iron Mountain Mine to the Sacramento River contributed to the listing of winter-run Chinook salmon as endangered (CVRWQCB 2002).

Current copper pollution from pesticides and urban runoff are not as extreme as the Iron Mountain Mine example; however, low levels of copper can have adverse effects on salmonids, other fish, invertebrates, and algae (Hecht et al. 2007; USEPA 2007). The most studied toxicity pathway of copper is its ability to disrupt ATP-driven pumps and ion channels, which results in impaired osmoregulation and ion regulation in gills (Kiaune and Singhasemanon 2011). However, fish sensory systems are likely the most sensitive to sub-lethal copper toxicity. For example, low-level copper exposures have been shown to disrupt

olfactory receptor neurons and lateral line mechanosensory neurons in fish (Hansen et al. 1999a; Hecht et al. 2007; Linbo et al. 2009; McIntyre et al. 2008; and Sandahl et al. 2007). In addition, these copper exposures resulted in measured behavior alterations (e.g., predator avoidance response, contaminant avoidance, and swimming) in Chinook salmon and rainbow trout that could result in reduced growth, survivability, and reproduction in salmonid populations (Hansen et al. 1999b; Sandahl et al. 2007).

Indirect Effects

Salmonid populations can also be adversely impacted indirectly by pesticides acting upon their target species. For example, herbicides and insecticides target the food web organisms that the salmonids depend on during rearing and migration. In addition, pesticides in the aquatic environment can shift algal or invertebrate communities to ones that are less nutritious or preferable to salmonids. Modifications to prey and prey food sources can have noticeable effects on fish populations (NMFS 2012). Reduced food for developing salmonids will result in greater competition, reduced fish growth, and possible starvation during critical life stages (NMFS 2008). Other possible indirect impacts to salmonid populations include the destruction of riparian vegetation (NMFS 2012). Riparian vegetation is important for providing shade, stabilizing stream banks, and providing allochthonous inputs that are important to maintaining salmonid ecosystems.

Population-level Effects

It is very difficult to quantify actual impacts that pesticide stressors have on salmonid populations because the effects can be direct or indirect, lethal or sublethal, long-term or short-term. To determine the possible combined effects that pesticides might have on salmon populations, researchers at the Northwest Fisheries Science Center used models to predict the effects of ChE inhibitors on anadromous Chinook salmon populations in the western United States (Baldwin et al. 2009; Macneale et al. 2014). They linked ChE activity to the somatic growth of subyearling Chinook salmon using a series of linear relationships (e.g., linked brain enzyme activity to feeding behavior, feeding behavior to food uptake, and food uptake to somatic growth). In addition, the researchers predicted the reduction in Chinook salmon growth due to reduced prey as a result of invertebrate exposure to pesticides. The predicted size of Chinook salmon at ocean entry is used to predict ocean survival, and then subsequent population growth.

The model results indicated that short-term exposures that were representative of real-world seasonal use patterns were enough to reduce the growth and size of juvenile Chinook salmon at the time of ocean entry. Consequently, the reduced size at ocean entry was enough to reduce the survival of individuals, which would, over successive years, reduce the intrinsic productivity of the population. For example, a four-day exposure to an organophosphate pesticide at a level that would produce a 50% reduction in ChE activity would result in a 6% decrease in the intrinsic population growth rate (Baldwin et al. 2009). Furthermore, the model estimated that if similar conditions continued for 20 years, then the exposed population spawner abundance would be only 27% of the unexposed spawner abundance. Macneale et al. (2014) evaluated additional pesticide classes (e.g., carbamates), exposure durations, and exposure frequencies. Overall, the magnitude of the responses indicates that common pesticides may significantly limit the conservation and recovery of threatened and endangered species in California (Baldwin et al. 2009).

Unfortunately, the models only evaluated the direct and indirect effects of single pesticide exposures at a time, and they did not incorporate possible interactions of multiple pesticides, other environmental stressors (e.g., reduced habitat and sub-optimal temperatures), or other contaminants. Different pesticides can work additively to cause a toxic effect, and other contaminants and stressors can influence pesticides' effectiveness, as well. For example, through transcriptional assays Hasenbein et al. (2014) determined that ammonia likely enhanced the effect of multiple-contaminant exposures to Delta smelt. Similarly, concurrent exposure of salmonids to copper and olfactory inhibitory pesticides could result in toxicological effects, even if both are at concentrations that would not elicit a response in isolation. Furthermore, many pesticides have been found to be able to work synergistically to cause toxicity to salmonids that is multiplicative and not just additive (Laetz et al. 2009). Current estimates of the effects of pesticides on salmonids may underestimate the true responses of salmonid populations in surface waters (Baldwin et al. 2009).

These additive and synergistic effects from multiple contaminants are true concerns for aquatic environments. For example, in the National Water-Quality Assessment (NAWQA) Program's monitoring of pesticides, they found that more than 90% of the streams located in developed areas contained two or more pesticides or degradates (Gilliom et al. 2006).

Furthermore, more than 50% of the streams had five or more pesticides or degradates, and the concentrations of the degradates were often higher than that of the parent pesticide. The degradate forms can be less toxic than the parent pesticide; however, some degradates have been found to be as toxic or more toxic than the parent (Gilliom et al. 2006). In addition, pesticide products typically contain additional chemicals like adjuvants, surfactants, and solvents. These chemicals are labeled as inert ingredients, but they increase the effectiveness of the active ingredients and can be toxic to non-target species (Beggel et al. 2010; Cox and Surgan 2006; and Scholz et al. 2012). Very little is known about the fate of these "inert" labeled ingredients once they are in surface waters and their possible impacts on salmonid populations.

1.3.1.2 Mercury

Mercury is a persistent and bioaccumulative toxic pollutant. Methylmercury is the most toxic form in the freshwater environment because it is the form most readily bioaccumulated in fish and through the food web (Wiener et al. 2003). For example, the proportion of mercury that exists as methylmercury generally increases with each level of the food chain, and methylmercury comprises 80% to 100% of the total mercury measured in fish tissue (Becker and Bigham 1995; Bloom 1992; Nichols et al. 1999; Slotton et al. 2004; and Sveinsdottir and Mason 2005; Weiner et al. 2003). Fish can absorb mercury through their epidermis (e.g., gills, skin) directly from water; however, fish accumulate the majority (greater than 85%) of their mercury through their diet in the form of methylmercury (Hall 1997; Weiner et al. 2003). There is evidence that methylmercury bioconcentrates (directly from water) in the laboratory (Fjeld et al. 1998; McKim et al. 1976); however, the minimum concentrations used in these dilution series exposures (160 nanograms per liter [ng/L] and 30 ng/L, respectively) were greater than 25-fold higher than the maximum aqueous methylmercury concentrations found in Central Valley mainstem rivers (Foe et al. 2008). It is the result of bioaccumulation and subsequent biomagnification that methylmercury concentrations typically become elevated in fish, and fish in the higher tropic levels tend to have the highest concentrations.

Fish have evolved in an environment that always contained mercury. Methylmercury is transported via the circulation system to all organs and tissue; however, methylmercury eventually redistributes to the skeletal muscles, where it becomes bound to proteins in the

muscle tissue (Weiner et al. 2003). In an extensive review of mercury impacts on fish, Weiner and Spry (1996) determined that the binding of assimilated methylmercury to proteins in the skeletal muscles may function as the primary detoxification mechanism for methylmercury in fish. The use of this mechanism reduces exposure of the central nervous system and brain to methylmercury. Because of the eventual redistribution of methylmercury to muscle tissue, the rate of accumulation and exposure time seem to significantly affect the toxicity of methylmercury to fish (Weiner and Spry 1996).

Neurotoxicity seems to be the most probable chronic response of wild fishes to dietary methylmercury, and long-term dietary exposure to methylmercury can cause incoordination, inability to feed, and diminished responsiveness (Weiner and Spry 1996). Other toxicological effects include reproductive impairments (e.g., hatching success, fecundity, and sex steroids), growth inhibition, developmental abnormalities (spinal and jaw deformities), altered behavioral responses (e.g., lethargy, predator response, and aggressiveness), and mortality (as reviewed in Beckvar et al. 1996; Beckvar et al. 2005; Depew et al. 2012; Dillon et al. 2010; Eisler 1987; Weis 2014; and Wiener and Spry 1996). Alterations in biochemistry, gene transcription, and tissue histology from exposure to mercury may also be the cause of the deleterious impacts to fish (Moran et al. 2007; Sandheinrich et al. 2011). For example, Moran et al. (2007) found differential gene expression in trout livers collected from two high elevation lakes in Washington. The fish collected from the more polluted lake, primarily higher mercury, exhibited upregulation of genes involved with a number of physiological processes including immune function, stress adaption, reproduction, and metabolism. Surprisingly, even the more contaminated lake fish had low levels of mercury contamination (less than 0.06 micrograms per gram [µg/g], wet weight, average of 2 years).

Mercury toxicity can have long lasting impacts well after exposure has ended. For example, Fjeld and others (1998) found that sub-lethal methylmercury exposures permanently impaired graylings (*Thymallus thymallus*) three years after the exposure. The 10-day egg exposures that resulted in embryo graylings tissue methylmercury concentrations of 3.8 µg/g (wet weight) exhibited immediate effects (e.g., delayed hatching, reduce hatching success, and malformed embryos); however, the embryos with body methylmercury concentrations as low as 0.27 µg/g exhibited reduce foraging success (e.g., feeding efficiency and competitive ability) compared to the control group three years after the initial methylmercury exposure.

Similarly, Matta and others (2001) observed transgenerational effects with killifish (*Fundulus heteroclitus*) fed methylmercury contaminated food. The maternal transfer of methylmercury to offspring resulted in altered sex ratios and other reproductive abnormalities in the next generation.

Reproductive and early life stage endpoints appear to be some of the most sensitive for fish species, and these adverse effects are typically seen at methylmercury tissue concentrations about 10-fold lower than seen for adult effects (Beckvar et al. 2005; Depew et al. 2012; Dillon et al. 2010; and Wiener and Spry 1996). Incubating salmonid eggs will be relatively unaffected by contaminants in the river because vitelline membrane, enveloping layer, and chorion provide defense from metals, pathogens, and xenobiotic chemicals (Finn 2007). Accordingly, the methylmercury accumulated in the eggs will be primarily derived from the maternal fish (Wiener and Spry 1996). Hammerschmidtt and Sandheinrich (2005) concluded that egg methylmercury was primarily derived from the maternal diet during oogenesis because offspring from adults fed mercury before and during oogenesis had similar concentrations as offspring from adults only fed during oogenesis; however, using stable isotope enriched methylmercury diets, Stefansson et al. (2014) found that both the maternal diet during oogenesis and the female tissue accumulated during preoogenesis contributed mercury proportionally to eggs.

The amount of methylmercury transferred from female to the egg appears to vary depending on contamination level, maternal length, species, etc. For example, the fathead minnow egg concentration percentages increased from 14% to 35% of maternal concentrations with increasing maternal methylmercury diets and maternal concentrations (Hammerschmidtt and Sandheinrich 2005). In another laboratory study with killifish, for the eggs that resulted in methylmercury concentrations above analytical detection limits the percentage of maternal muscle methylmercury concentration in eggs was 0.9% and 5.3%, also increasing with dosage and maternal concentration (Matta et al. 2001). In a field investigation, Johnston et al. (2001) found that egg methylmercury concentrations were 1.1% to 12% of female muscle concentrations for seven different walleye (*Stizostedion vitreum*) populations. In addition, the percentage of the maternal concentrations varied with maternal length, egg concentrations, maternal liver and muscle concentrations, female length, and population location. Finally, Niimi (1983) investigated the maternal transfer of multiple contaminants

in five different species collected from Lake Ontario and Erie. The percentage of maternal methylmercury concentrations in eggs averaged: 0.6% for rainbow trout (*O. mykiss*), 1.8% for white sucker (*Catostomus commersoni*), 0.3% for white bass (*Morone chrysops*), 0.4% for smallmouth bass (*Micropterus dolomieui*), and 2.3% for yellow perch (*Perca flavescens*). The field investigations are likely most indicative of typical maternal transfer to eggs from the natural environment because these fish reflect the natural bioaccumulation rates, prey methylmercury concentrations, and growth rates.

1.3.1.3 Selenium

Selenium is an essential micronutrient for normal animal nutrition; however, selenium can bioaccumulate and biomagnify to levels that are toxic to fish and other wildlife. Selenium can bioconcentrate directly from water through gills, epidermis, or gut; however, like mercury, the primary route of exposure to levels that exhibit toxicological effects is through the food web (Hamilton 2004; Lemly and Smith 1987; Presser and Luoma 2013; USEPA 2014; and Entrix 2009). When dissolved selenium enters the aquatic environment, it may do the following (Lemly and Smith 1987):

- Be absorbed or ingested by organisms
- Bind or complex with particulate matter
- Remain in solution

The speciation of dissolved selenium in its three dominant oxidation states (i.e., selenate, selenite, or dissolved organic selenium) is important because the oxidation state of the dissolved form influences the rate of transformations (e.g., oxidation and methylation) that create the particulate form (Lemly and Smith 1987; Presser and Luoma 2013). The uptake of selenate by plants and phytoplankton appears to be slower than the other two oxidation states (Presser and Luoma 2013).

Ecologically, the first and second mechanisms above are the most important because particulate selenium and selenium associated with plants and phytoplankton are the primary forms that enter the food web (Lemly and Smith 1987; Presser and Luoma 2013; USEPA 2014). Examples of the mechanisms where selenium is made available for biological uptake include the following (Lemly and Smith 1987; Presser and Luoma 2013):

- Oxidation and methylation of inorganic and organic selenium by plant roots and microorganisms
- Biological mixing and associated oxidation of sediments that results from the burrowing of benthic invertebrates and feeding activities of fish and wildlife
- Physical perturbation and chemical oxidation associated with water circulation and mixing
- Oxidation of sediments by plant photosynthesis
- Recycling of particulate phases back into water as detritus or dissolved organic selenium after organisms die and decay

In addition, rooted plants and detrital feeding organisms can input selenium into the food web, even when selenium is absent from the water column (Lemly and Smith 1987).

Selenium has three levels of biological activity in fish: 1) trace concentrations are required for normal growth and development, 2) moderate concentrations can be stored and homeostatic functions maintained, and 3) elevated concentrations can result in toxic effects (Hamilton 2004). Fish exposure to selenium typically follows a biphasic response (i.e., beneficial at low doses and toxic at high doses [USFWS 2008; Hilton et al. 1980; and Lemly and Smith 1987]). Toxic effects of selenium to fish typically fall into two categories (Lemly and Smith 1987; USEPA 2014):

- Chronic reproductive (e.g., effects to offspring survival and morphology)
- Chronic non-reproductive (e.g., adult and juvenile growth and survival)

Similar to mercury, reproductive function is the most sensitive to selenium toxicity, and the most documented impacts to reproduction are teratogenesis and larval mortality (USEPA 2014). Often, reproductive failure, whether through effects on adult ovaries or embryonic development, are the first obvious symptom of selenium contamination, and complete reproductive failure can occur with very little or no tissue pathology or mortality of the adult population (Lemly and Smith 1987). USFWS' (2008) review of selenium impacts to threatened and endangered species in the Delta reported statistically significant increases in pre-swimup mortality and increased percentages of edema and craniofacial deformities in swimup fry with increasing egg selenium concentrations in rainbow trout. In addition, others have reported that fish exposed to selenium exhibit ovaries with necrotic and

ruptured egg follicles, anemia and reduce hatch in eggs, or chromosomal aberrations (Eisler 1985). Additional effects of selenium to early life stage fish include deformities that include: lordosis (concave curvature of lumbar and caudal regions of spine), kyphosis (convex curvature of thoracic region of the spine), scoliosis (lateral curvature of the spine); in addition to edema, and brain, heart, and eye problems (Hamilton 2004).

Selenium is transferred from the maternal diet to developing eggs during vitellogenesis, and the embryo is exposed to selenium during yolk absorption (Presser and Luoma 2013; USEPA 2014). The rate of maternal transfer of selenium to gonadal tissue is much greater than for mercury. For example, Linares-Casenave et al. (2014) found that white sturgeon (*Ancipenser transmontanus*) sampled from the San Francisco Bay and Delta had gonadal tissue selenium concentrations 100 and 200% that of muscle selenium concentrations in previtellogenic and vitellogenic females, respectively. This is compared to the maternal transfer of 0.3% to 12% of mercury concentrations in gonadal tissues observed in field collected fish (see above). For the development of their draft Aquatic Life Ambient Water Quality Criterion for Selenium, USEPA (2014) summarized paired maternal and egg-ovary selenium concentrations to estimate conversion factors between tissue concentrations. Individual species conversion factors (maternal muscle>egg-ovary) ranged from 1.0 to 5.8 (i.e., egg concentrations were 100% to 580% of maternal concentrations), with rainbow trout having the second highest transfer rate (out of 16 species) with a conversion factor of 1.9. The overall high ranking of salmonids continued at the genus level (average *Oncorhynchus* = 1.9) and family level (average *Salmonidae* = 1.5).

Beyond the reproductive and early life stages, additional effects can occur in fish at later exposures. For example, juvenile rainbow trout fed selenium supplemented diets exhibited reduce growth, higher feed:gain ratio, and higher number of mortalities after 20 weeks of feeding (Hilton 1980). In addition, the juveniles exhibited behavior effects (e.g., feeding avoidance) as well as uncoordinated swimming and sensory deprivation approximately 24 hour priors to mortality. Similarly, Hamilton and Wiedmeyer (1990) found that reduced survival and growth of Chinook salmon were strongly correlated to tissue selenium concentrations in 90-day exposures. As well, selenium exposures to Chinook salmon resulted in reduced survival in the 15-day seawater challenge. Additional effects to fish include: loss of equilibrium, lethargy, contraction of dermal chromatophores, loss of coordination, muscle

spasms, protruding eyes, swollen abdomen, liver degeneration, reduction in blood hemoglobin and erythrocyte number, increase in white blood cells, and swollen gill lamellae with extensive cellular vacuolization (Eisler 1985).

In addition to being an essential micronutrient for organisms, selenium has been found to have protective effects against mercury and other metal toxicity (Eilser 1987; USEPA 2014). However, the mechanism for the antagonistic interactions is not known, the degree of antagonism is highly variable, and some studies found additive and synergistic interactions with mercury. Laboratory studies by Bjerregaard et al. (2011) suggested that selenium increases the elimination of methylmercury in fish; however, the report acknowledges that other have suggested that selenium may reduce mercury toxicity by redistributing mercury to different tissues or by reducing the assimilation of mercury. Regardless of the mechanism, selenium availability (excess and deficiency) in the aquatic ecosystem must be considered, when considering optimal concentrations in the environment.

1.3.2 Approach

1.3.2.1 Pesticides

The SEP group relied on adopted numeric water quality objectives for pesticides from the Sacramento and San Joaquin River Water Quality Control Plan, and proposed pesticide water quality objectives from developing pesticide control programs (CVRWQCB 2011, 2014, 2015) to determine pesticide levels that should provide no adverse impacts to adult migration. In addition, for pesticides that do not have state or federally promulgated objectives or criteria, the SEP group used the USEPA Office of Pesticide Programs (OPP) aquatic-life benchmarks with a level of concern for impacts to endangered and threatened species as the safe level for pesticides.

Unfortunately, no pesticide monitoring program exists throughout Stanislaus River, San Joaquin River, Delta or Bay, nor is there likely a program that will exist in the future that will be able to monitor all possible pesticides that may adversely impact salmonids during entire life stages. Quantifying the concentrations of all the pesticides that salmonids are exposed to is difficult. For example, more than 1,000 pesticide chemicals were applied in California in 2012 (CDPR 2014). In addition, each commodity or crop type can have multiple pesticide chemicals that are applied to them (e.g., alfalfa crops were associated with

greater than 200 pesticide chemicals). Performing chemical analyses, for all possible pesticides in the different reaches of the river where salmonids would be exposed, would not be cost feasible. Furthermore, current analytical methodologies do not allow for all pesticides to be detected at levels that may cause adverse effects to aquatic organisms (CVRWCB 2015; Hladik et al. 2009; Mekebri 2011).

Additionally, each of the specific pesticides has different impacts to the physiology of salmonids, as well as to their prey. For example, Macneale et al. (2014) population modeling determined that the magnitude of a pesticide's effect on salmon population growth is dependent on the relative sensitivity of salmon olfactory senses and prey abundance to the pesticide. For instance, chlorpyriphos had a greater influence on salmon population growth by directly affecting salmon physiology, while another organophosphate, diazinon, had a greater impact by decreasing salmon prey abundance. Attempting monitor and evaluate the direct and indirect effects of the more than 1,000 possible pesticides and mixtures of pesticides that could occur in the Stanislaus River and downstream corridor would very difficult.

The SEP group has relied on a pesticide prediction model (Hoogeweg et al. 2011) to estimate the current frequency of pesticide water quality objective or benchmark exceedances to categorize optimal, sub-optimal, and detrimental conditions for Chinook salmon and steelhead pesticide environmental objectives. That is, the categories are an evaluation of the risks that a species is exposed to pesticide concentrations that could cause harm in a river reach by month. The categories assume that, while zero occurrences of pesticides is preferred, such low levels of exposure may not be achievable considering the amount of urban and agricultural development in the Central Valley. Models, monitoring, toxicity bioassays, and other information, will need to be updated, developed, conducted, and further gathered as needed in the future to determine if pesticide concentrations are adversely impacting salmonids in through their life stages.

1.3.2.2 Mercury

Current mercury numeric water quality objectives or criteria were developed to protect human and other fauna that consume fish and not for the protection of fish themselves. For example, the USEPA-promulgated California Toxics Rule (CTR) numeric criteria for mercury

is for the protection of human health only (40 Code of Federal Regulations [CFR] Part 131). As noted earlier, fish with elevated concentrations of mercury are frequently observed in waterbodies that do not exceed the CTR criterion of 0.05 micrograms per liter (µg/L) total mercury (Wood et al. 2010). Similarly, water quality objectives developed individually for the San Francisco Bay and the Delta were developed as fish tissue objectives for the protection of human and wildlife consumers of fish (Wood et al. 2010; SFBWQCB 2006). This is in part due to the fact that until recently (within the last decade), the majority of evidence supported that fish were relatively insensitive to mercury toxicity when compared to human and wildlife consumers of fish (Weiner and Spry 1996). For example, Wiener and Spry (1996) concluded that estimated no-observed-effect mercury concentrations for salmonids were 3 µg/g (wet weight, whole body), whereas fish tissue mercury concentrations to protect human and wildlife consumers of fish from the San Francisco Bay and Delta is greater than 10-fold lower at approximately 0.2 µg/g (wet weight, muscle tissue[2]) (Wood et al. 2010; SFBWQCB 2006).

Since 1996, many studies have reported adverse effects to fish species at concentrations lower than the papers reviewed by Wiener and Spry, and there is now evidence that fish species are more sensitive to mercury toxicity than previously thought (Dillon et al. 2010). For example, Beckvar et al. (2005) developed approaches (i.e., simple ranking, empirical percentile, tissue threshold-effect level (t-TEL), and cumulative distribution function) to determine the fish tissue mercury concentrations that would be protective against adverse mercury toxicity using studies that measured mercury tissue concentrations and corresponding biological responses (e.g., reproduction, growth, and behavior) in adult, juvenile eggs, and early-life stages (ELS) fish. Dillon et al. (2010) used dose-response curves on lethality-equivalent test endpoints to estimate the percent injury to fish by mercury. The SEP group relied in these benchmark concentrations as the levels that would be optimal, sub-optimal, and detrimental to salmonids during their life stages.

1.3.2.3 Selenium

The SEP group relied on the draft USEPA National Freshwater Selenium Ambient Water

[2] Muscle tissue (filet) mercury concentrations can be converted to whole-body mercury (Hg) concentrations using the following equation: Log [filet biopsy Hg] = 0.2545 + 1.0623 x Log [whole-fish Hg] (Peterson et al. 2007).

Quality Criterion for Aquatic Life (2014) for the environmental objectives to protect salmonid species in the Stanislaus River against adverse effects. The criteria have yet to be promulgated; however, the criteria are consistent with the relevant technical literature on selenium toxicology.

1.3.3 Objectives

Some of the identified contaminants have associated USEPA promulgated numeric aquatic life water quality or human health criteria (CTR, 40 CFR Part 131) as well as each may have Regional Board specific water quality objectives. Unfortunately, most current use pesticides do not have promulgated water quality criteria or objectives. Additionally, the CTR criteria were developed to protect for human health or against short-term (4-day) effects on aquatic life, and these criteria may not be protective of long-term (e.g., weeks, months, and years) adverse impacts on salmonids and other wildlife. For example, the evaluation for the Sacramento-San Joaquin Delta Estuary Total Maximum Daily Load (TMDL) for methylmercury determined that even though the CTR criterion for mercury is never exceeded in the Delta, fish tissue mercury concentrations are a threat to threatened and endangered wildlife species and humans that consume Delta fish (Wood et al. 2010). As well, many of the toxicological studies to be discussed later have observed adverse effects to salmonids below established water quality criteria.

1.3.3.1 Pesticide Objectives

Numeric water quality objectives have not been established for vast majority of current use pesticides in the Central Valley. Table B-11 presents the pesticides that have adopted numeric water quality objectives in the Sacramento and San Joaquin River Basins Water Quality Control Plan (Basin Plan) and the proposed water quality objectives for pyrethroid pesticides (CVRWQCB 2011, 2014, and 2015).

Table B-11
Central Valley Regional Water Quality Control Board Adopted and Proposed Water Quality Objectives for Current Use Pesticides

Pesticide	Acute (µg/L)	Chronic (µg/L)
Adopted Water Quality Objectives[1]		

Diazinon	0.16	0.1
Chlorpyriphos	0.025	0.015
Carbofuran	40	40
Simazine	4	4
Thiobencarb	1	1
Pentachlorophenol	5.3	4
Copper	5.7	4.1
Proposed Water Quality Objectives[2]		
Bifenthrin	0.00006	0.00001
Cyfluthrin	0.0002	0.00004
Lambda-Cyhalothrin	0.00003	0.00001
Cypermethrin	0.00004	0.00001
Esfenvalerate	0.0002	0.00003
Permethrin	0.006	0.001

Notes:
[1] CVRWQCB 2011
[2] Proposed water quality objectives for the Central Valley Pyrethroid Pesticides Total Maximum Daily Load (TMDL) and Basin Plan Amendment (CVRWQCB 2015).
µg/L = microgram per liter

USEPA OPP develops aquatic toxicity benchmarks for use in risk assessment and pesticide registration decisions under the Federal Insecticide, Fungicide, and Rodenticide Act (USEPA 2004). OPP has developed aquatic life benchmarks for over 400 registered pesticides. Table B-12 presents the benchmarks for the 40 pesticides that are predicted to pose the greatest risks in the Central Valley (Lu and Davis 2009; Hoogeweg et al. 2011). Included in Table B-12 are the benchmarks for the protection of the critical habitat for listed species, which includes an additional safety factor (USEPA 2004). The aquatic life benchmarks can be used for initial environmental assessments; however, a more detailed evaluation or site-specific evaluations may determine that the aquatic life benchmarks are not protective of the most sensitive species. For example, a comparison between the OPP benchmarks (Table B-12) and the established or proposed water quality objectives (Table B-11) shows that all but one of the water quality objectives predicts that a lower concentration than the OPP benchmarks is necessary to protect beneficial uses. Attaining the lower of either the aquatic life benchmarks or the water quality objectives should reasonably allow for the protection of salmonid species as well as their habitat.

Table B-12

USEPA Office of Pesticide Programs' Aquatic-Life Benchmarks for the 40 Pesticides that Pose the Greatest Risk in the Central Valley Region

Pesticide	Pesticide Type	Acute Benchmark (µg/L)	Endangered and Threatened Acute Benchmark (µg/L)	Chronic Benchmark (µg/L)	Source of Acute/ Chronic Value[1]
Abamectin	Insecticide	0.17	0.017	0.006	IA/IC
Bifenthrin	Insecticide	0.075	0.0075	0.0013	FA/IC
Bromacil	Herbicide	6.8	0.68	3000	AA/FC
Captan	Fungicide	13.1	1.31	16.5	FA/FC
Carbaryl	Insecticide	0.85	0.085	0.5	IA/IC
Chlorothalonil	Fungicide	1.8	0.18	0.6	IA/IC
Chlorpyrifos	Insecticide	0.05	0.005	0.04	IA/IC
Clomazone	Herbicide	167	16.7	350	AA/FC
Copper hydroxide	Fungicide	5.9	0.59	4.3	IA/IC
Copper sulphide	Insecticide/Algaecide	5.9	0.59	4.3	IA/IC
Cyfluthrin	Insecticide	0.0125	0.00125	0.007	IA/IC
Cyhalofop butyl	Herbicide	245	24.5	134	FA/FC
Cypermethrin	Insecticide	0.195	0.0195	0.069	FA/IC
Deltamethrin	Insecticide	0.055	0.0055	0.0041	IA/IC
Diazinon	Insecticide	0.11	0.011	0.17	IA/IC
Dimethoate	Insecticide	21.5	2.15	0.5	IA/IC
Diuron	Herbicide	2.4	0.24	26	AA/FC
Esfenvalerate	Insecticide	0.025	0.0025	0.017	IA/IC
Hexazinone	Herbicide	7	0.7	17000	AA/FC
Imidacloprid	Insecticide	35	3.5	1.05	IA/IC
Indoxacarb	Insecticide	12	1.2	3.6	FA/IC
Lambda cyhalothrin	Insecticide	0.0035	0.00035	0.002	IA/IC
Malathion	Insecticide	0.3	0.03	0.035	IA/IC
Mancozeb	Fungicide	47	4.7	N/A	AA/na
Maneb	Fungicide	13.4	1.34	N/A	AA/na
Methomyl	Insecticide	2.5	0.25	0.7	IA/IC
(s)-Metolachlor	Herbicide	8	0.8	30	AA/FC
Naled	Insecticide	25	2.5	0.045	AA/IC
Oxyfluorfen	Herbicide	0.29	0.029	1.3	AA/FC
Paraquat	Herbicide	0.396	0.0396	N/A	AA/na

Pendimethalin	Herbicide	5.2	0.52	6.3	AA/FC
Permethrin	Insecticide	0.01	0.001	0.0014	IA/IC
Propanil	Herbicide	16	1.6	9.1	AA/FC
Propargite	Insecticide	37	3.7	9	IA/IC
Pyraclostrobin	Fungicide	0.0015	0.00015	0.002	FA/FC
Simazine	Herbicide	36	3.6	960	AA/FC
Thiobencarb	Herbicide	17	1.7	1	AA/IC
Tralomethrin	Insecticide	0.055	0.0055	0.0041	IA/IC
Trifluralin	Herbicide	7.52	0.752	1.14	AA/FC
Ziram	Fungicide	9.7	0.97	39	FA/IC

Notes:
Source: USEPA Office of Pesticide Program (OPP)
Table modified from Hoogeweg et al. (2011).
Aquatic-life benchmarks are used by the USEPA-OPP for risk assessments in the registration of pesticides. To assess a pesticide not listed, the entire list of nearly 500 pesticide benchmarks can be acquired at: http://www.epa.gov/oppefed1/ecorisk_ders/aquatic_life_benchmark.htm
[1] Identifies which taxa was the most sensitive to the pesticide from available toxicity evaluations: FA = fish acute; IA = invertebrate acute; AA = Algae Acute; FC = fish chronic; IC = invertebrate chronic; na = not available
µg/L = microgram per liter

The pesticide criteria and benchmarks were developed assuming organismal exposure to single pollutants. Additional considerations are necessary, when multiple pesticides are present (e.g., additive toxicity equations) (CVRWQCB 2011, 2014, 2015; Hasenbein et al. 2014). In addition, assessing the true impact on aquatic life may need to consider the bioavailability of the pesticides (CVRWQCB 2015). For example, the majority of dissolved copper is likely bound as ligand complexes and largely not bioavailable (Linbo et al. 2009; McIntyre et al. 2008; SFBRWQCB 2007). Consequently, copper, pesticides, and other metals toxicity evaluations should involve adjustments for site-specific conditions (e.g., hardness, biotic ligand models, or dissolved organic concentrations) (CVRWQCB 2014, 2015; SFBRWQCB 2007).

The Hoogeweg et al. (2011) model allowed the determination of the magnitude of pesticide effects on Stanislaus River salmonids, and the relative risk of pesticide exposures by month and river reach (Figure B-1 and Table B-13). As mentioned earlier, limitations in monitoring and chemical analyses, the multitude of possible pesticide chemicals, etc. precludes the use of strict concentration limitations to evaluate overall pesticide impacts on salmonids throughout the Stanislaus River and downstream waterbodies. In turn, current pesticide impacts to

salmonid life stages in the Stanislaus River are based on the relative frequency of pesticides exceeding aquatic-life benchmarks.

Table B-13
Categories of Predicted Pesticide Aquatic-life Benchmark Exceedances

Bin Category	Condition	Range of the Frequency of Benchmark Exceedances		
1	Optimal	0	-	0.017
2	Sub-optimal	0.018	-	0.055
3		0.056	-	0.1
4		0.101	-	0.153
5		0.154	-	0.206
6		0.207	-	0.303
7	Detrimental	0.304	-	0.447
8		0.448	-	0.5
9		0.501	-	0.589
10		0.59	-	0.994

Note:
Frequencies were calculated from the total number of predicted exceedance days for each month from 2000 to 2009. Any day that had at least one pesticide that exceeded benchmarks was counted as an exceedance day (adapted from Hoogeweg et al. 2011).

To be fully protective of aquatic-life beneficial uses, current pesticide water quality objectives and criteria require that pesticide thresholds are not exceeded more than once every 3 years (40 CFR Part 131; CVRWQCB 2014). Similarly, meeting the frequency range of Bin 1 (Table B-13) of pesticide exposure in the Stanislaus River and freshwater migratory corridor should allow the full expression of salmonid life stages, and this represents the optimal condition. Furthermore, the analysis for the development of the Central Valley diazinon, chlorpyriphos, and pyrethroid TMDLs concluded that the adopted and proposed numeric criteria for these pesticides should be reasonably achievable (CVRWQCB 2014, 2015).

Determining the frequency of pesticide exposures that are predicted to result in sub-optimal versus detrimental impacts is much more difficult. For example, as mentioned previously the Northwest Fisheries Science Center modeling determined that the effect of pesticides on the intrinsic population growth of salmon was dependent on the relative sensitivity of salmon

olfactory function versus prey abundance to specific pesticides, the binding affinity of specific pesticides, the concentration of pesticide in the habitat, and the duration and frequency of pesticide exposures (Baldwin et al. 2009; Macneale et al. 2014). However, overall the models predicted that the impact to prey abundances had a greater effect on the salmon intrinsic population growth than the direct physiological effects to salmon with regards to juvenile growth.

A single 4-day pulse of high pesticide concentrations (e.g., 1.15 x prey abundance EC50 or 60-fold acute WQO) resulted in a 1% to 11% reduction in salmonid population growth depending on prey recovery rates (Macneale et al. 2014). In terms of spawner abundance, a 1% and 7% decrease in intrinsic population growth would equate to a 14% and 73%, respectively, reduction in spawner abundance compared to an unexposed control after 20 years (Baldwin et al. 2009). However, this high concentration of pesticides is at the upper range of pesticides observed in salmonid habitats and may not represent typical conditions (Baldwin et al. 2009). Fortunately, the researchers modeled a continuous low pesticide concentration exposure (e.g., salmon olfaction inhibition EC10 or 6-fold acute WQO), which lasted 105 out of 140 or 75% of the modeled rearing period. The estimated reduction in population growth was 4% or a 53% reduction in spawner abundance after 20 years.

A 4% reduction in intrinsic population growth or 75% frequency of pesticide exposure would likely still represent detrimental conditions to salmonid populations; however, a 2% reduction in intrinsic population growth (e.g., 1.08 versus the 1.1 control population) would likely represent conditions where salmonid populations are impacted but can still attain biological objectives. Accordingly, Bin 7 or greater (Table B-13), which represent approximately one-half of the 75% exposure frequency and greater, is considered to be detrimental to salmonid populations. These reductions in population growth were through impairments in salmon olfaction. The SEP group assumes that the degree of olfaction disruption would have equivalent impact on overall fitness during each life stage.

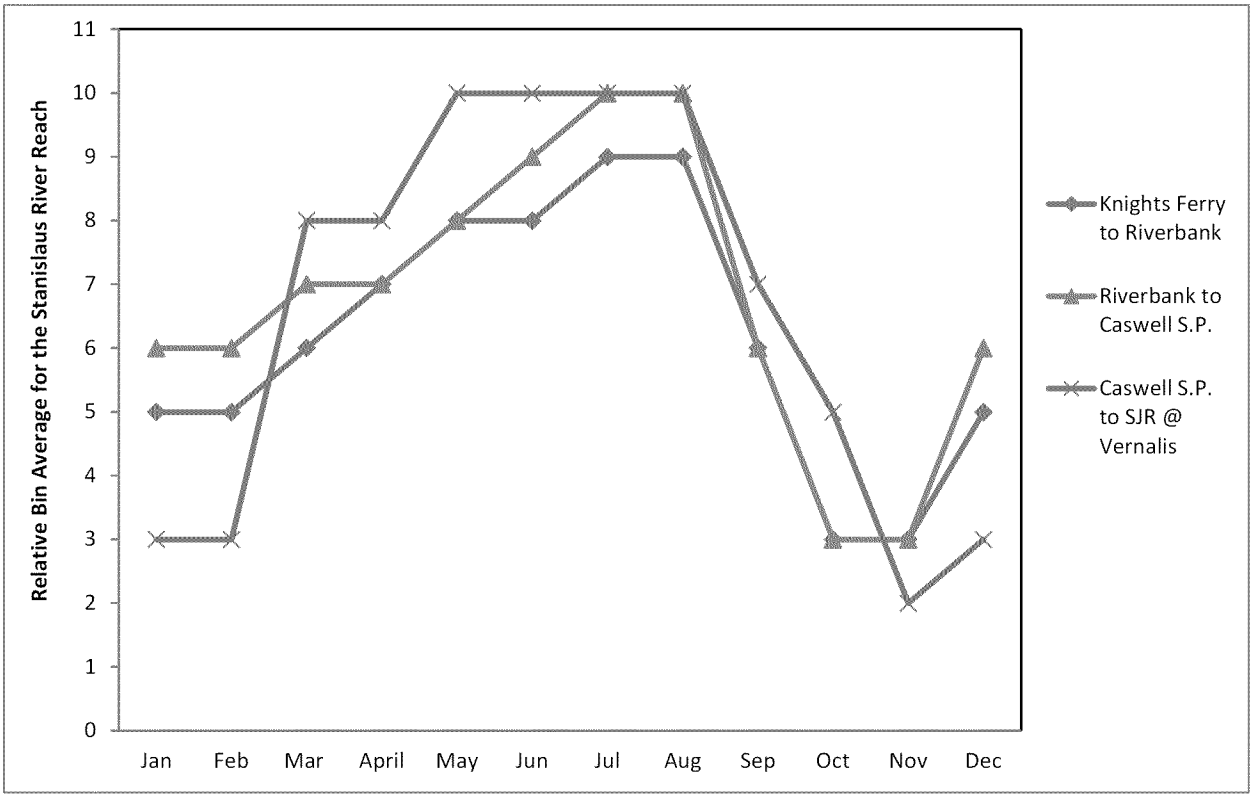

Figure B-1

Relative Bin Value of Specified Stanislaus River Reaches by Month

Note:
The values were derived from qualitative averaging of the frequency of benchmark exceedances model maps for years 2000 to 2009 in Hoogeweg and others (2011). Due to a lack of data, upstream of Knights Ferry in the Stanislaus River was not modeled.

1.3.3.2 Mercury Objectives

Using the methodology described in Section 1.3.2, a whole-fish mercury concentration of 0.2 µg/g (wet weight) (filet = 0.33 µg/g, wet weight) is predicted to be protective of juvenile and adult fish using the t-TEL method. Using the simple ranking method, Beckvar et al. (2005) estimated that 0.02 µg/g whole-body would be protective of early-life stage fish, which is consistent with the hypothesized higher sensitivity of sublethal effects to embryonic and larval stages mentioned earlier. These values are consistent with the percent of injury to fish by mercury estimate by Dillon et al. (2010) using dose-response curves on lethality-equivalent test endpoints. Sub-optimal and detrimental conditions are displayed in Table B-14. Both Beckvar et al. (2005) and Dillon et al. (2010) developed the fish mercury concentration thresholds using multiple species; however, these thresholds should also be

protective of salmonids because the development of the thresholds considers the most sensitive species and endpoints. In addition, there is evidence that salmonid species are less sensitive to the toxicity of dietary methylmercury (Berntssen et al. 2004 as cited in Depew et al. 2012).

Table B-14
Mercury Objectives for Chinook Salmon and Steelhead for Juveniles and Adults and Egg, Ovary, and Early-Life Stages

Condition	Egg/Ovary/ELS mg/kg (wet wt.)	Adult and Juvenile Fish mg/kg whole body (wet wt.)
Optimal	< 0.02	< 0.20
Sub-optimal	0.02 to 0.10	0.20 to 1.0
Detrimental[1]	> 0.1	> 1.0

Notes:
[1] Sub-lethal impacts to fish are estimated to occur above optimal conditons. Detrimental impacts are assumed to occur at mercury tissue concentrations that are expected to create 25% or greater injury to the fish. A 25% effect or EC25 metric is a consistent threshold to determine chronic toxicity assessments for regulatory compliance (SWRCB 2012).
">" = greater than
"<" = less than
ELS = early-life stages
mg/kg = milligram per kilogram
wt. = weight

1.3.3.3 Selenium Objectives

USEPA reserved the aquatic life criteria for selenium in the CTR because a USFWS and National Marine Fisheries Service (NMFS) biological opinion found that the proposed criteria for selenium may not be protective for threatened and endangered species (USFWS and NMFS 2000). In 2014, USEPA drafted proposed selenium ambient chronic water quality criteria for the protection of aquatic life (Table B-15). The proposed criterion allows for multiple matrices to be evaluated (e.g., egg/ovaries, adult fish, and water); and, it takes into consideration that reproduction and early-life stages are the most sensitive to selenium toxicity. In addition, the criterion defaults to tissue selenium concentrations over aqueous selenium concentrations because aqueous concentrations may not reflect the principal exposure routes (e.g., food web and maternal transfer) (Entrix 2009; USEPA 2014).

Table B-15

USEPA Draft National Freshwater Selenium Ambient Water Quality Criterion for Aquatic Life

Media Type	Fish Tissue		Water Column	
Criterion Element	Egg/Ovary	Fish Whole Body or Muscle	Monthly Average Exposure	Intermittent Exposure
Magnitude	15.2 mg/kg (dry wt.)	8.1 mg/kg whole body or 11.8 mg/kg muscle (skinless, boneless filet) (dry wt.)	1.3 µg/L in lentic aquatic systems 4.8 µg/L in lotic aquatic systems	$WQC_{int} = \frac{WQC_{30\text{-}day} - C_{bkgrnd}(1 - f_{int})}{f_{int}}$
Duration	Instantaneous measurement	Instantaneous measurement	30 days	Number of days/month with an elevated concentration
Frequency	Never to be exceeded	Never to be exceeded	Not more than once in 3 years on average	Not more than once in 3 years on average

Notes:
From USEPA 2014. These draft criteria are presented to give a relative magnitude of selenium levels above which could pose risks to aquatic life. In addition, the criteria are presented as an example of the type of approach that could be used to assess selenium impacts to aquatic life. The criteria have yet to be peer review, and they have not been promulgated by USEPA.
µg/L = microgram per liter
mg/kg = milligram per kilogram
WQC = Water Quality Criterion
wt. = weight

The proposed draft criterion for selenium is similar to other criteria and levels of concern determined by others. For example, the CVRWQCB water quality objectives for selenium are 5 µg/L and 2 µg/L in the San Joaquin River and Salt Slough, respectively. The draft USEPA aquatic life criterion presents 2 different concentrations because it considers the differences in selenium exposure and bioaccumulation rates of lentic and lotic systems. Based on laboratory toxicity tests, Hamilton and Wiedmeyer (1990) suggested that adverse effects for could occur between 3 and 5 µg/g in young salmon (5 g or less) and between 4 and 8 µg/g in older salmon (18 g or more). In a later review by Hamilton (2004), the author reported that no effects were typically not observed below 4 µg/g (whole body, dry weight) and suggested that the majority of the literature supports threshold starting around 4 µg/g.

USFWS (2008) developed statistical models and predicted that 2.5 µg/g would result in a 20% effect in mortality in juvenile Chinook salmon and 2.15 µg/g would result in a 20% reduction

in growth in juvenile rainbow trout. However, the data used to develop this model was found to be unacceptable for USEPA to use for the development of criteria (USEPA 2014). There appears to be some uncertainty in the levels of selenium that adversely impact salmonid growth. The environmental objective for selenium in the Stanislaus River should be re-evaluated, once the USEPA finalizes its criteria or studies reduce the uncertainty in selenium toxicology.

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